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## MEETING REPORT

# Meeting report – Cell dynamics: organelle–cytoskeleton interface

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### ABSTRACT

A hallmark of eukaryotic cells is the spatial separation of molecular and biochemical processes into membrane-bound organelles, such as mitochondria, endoplasmic reticulum and Golgi. At the ‘Cell dynamics: organelle–cytoskeleton interface’ meeting held in Lisbon, researchers from around the world discussed their findings of how the cytoskeleton regulates dynamics, interaction, and function of organelles in health and disease. Organised by Edgar Gomes, Heidi McBride, Sharon Tooze and Michael Way, the meeting created an open, stimulating and collaborative environment for scientific exchange and an opportunity to highlight the newest trends in the field.

### Introduction

An emerging theme in cell biology is the functional importance of organelle positioning and connectedness, and its regulation by the cytoskeleton. In the opening keynote lecture, Pietro de Camilli (Yale University and HHMI, USA) presented the role of VPS13 proteins at contact sites of the endoplasmic reticulum (ER) with mitochondria (VPS13A), and of the ER with late endosomes/lysosomes (VPS13C). Pietro described that the N-terminal region of VPS13 proteins acts as a lipid transport module for glycerolipid transport at organelle contact sites (Kumar et al., 2018), and suggested that VPS13A in mammalian cells may fulfil some of the functions of the well-studied yeast ERMES complex. Notably, the importance of inter-organelle contact sites is indicated by the role of VPS13A and VPS13C mutations in neurodegenerative diseases, such as chorea acanthocytosis (VPS13A) and Parkinson’s disease (VPS13C).

In the second keynote lecture, Jennifer Lippincott-Schwartz (HHMI Janelia Research Campus, Ashburn, USA) investigated inter-organelle contact sites (the ‘organelle interactome’) in a systematic approach by using lattice light sheet multispectral imaging (Valm et al., 2017) and also focussed ion beam milling combined with scanning electron microscopy (FIB-SEM) in combination with machine learning approaches to overcome the light diffraction limit. Jennifer further approached the exciting question of how membrane-lacking liquid granules are transported within the cell, and how dysfunction might be associated with neurodegenerative diseases. Using the transport of RNA granules in axons as a model, her group discovered that RNA granules co-traffic with lysosomes by the linker annexin A11, which contains annexin repeats to connect to lysosomes and an intrinsically disordered domain to connect to RNA granules.

Both keynote lectures perfectly represented the general trend in the field to advance our scientific knowledge by technical advances

[such as correlative light and electron microscopy (CLEM) and live-cell high-resolution microscopy], by studies in differentiated cell types (such as neurons), and by exploring the consequences of dysfunction (such as in neurodegenerative diseases).

### Mitochondrial fission, movement and function

Regulation of mitochondrial dynamics and function is essential for meeting cellular energy demands. Heidi McBride (McGill University, Montreal, Canada) kicked off this theme with live imaging data that showed a new role for PI4P in driving mitochondrial division at sites of inter-organelle contact (Sugiura et al., 2014). This was followed by a fascinating talk from Benoît Kornmann (University of Oxford, UK) who inadvertently identified that CENP-F, a kinetochore protein, is also a mitochondrial protein. Live imaging experiments revealed that CENP-F is recruited to mitochondria in G2 and interphase by Miro1. This interaction is important for mitochondrial distribution – a single amino acid change in the Miro–CENP-F interaction region, introduced through CRISPR, results in mitochondrial clumping at the nucleus. Based on data from super-resolution imaging in cells and TIRF microscopy in *in vitro* reconstitution assays, he argued for a model whereby CENP-F may not be a walking motor, but rather a microtubule-binding mitochondria mobiliser (Peterka and Kornmann, 2019).

Vaishnavi Ananthanarayanan (Indian Institute of Science, Bengaluru, India) then addressed the function of fission yeast microtubules in mitochondrial fission. She demonstrated that the association of microtubules with mitochondria blocks their fission and that inhibition of the mitochondria–Dnm1 interaction underpins this. She suggested that during cell division, dissociation of mitochondria from microtubules and their subsequent fragmentation are necessary to ensure correct partitioning between daughter cells (Mehta et al., 2019). The next talk by Vidhya Rangaraju (MPI for Brain Research, Frankfurt, Germany) sought to illuminate how localised protein synthesis is fuelled at postsynaptic sites of neurons. She demonstrated that this energy need is met by oxidative phosphorylation in mitochondria, which are organised in distinct and stable compartments. This compartmentalisation is achieved by cytoskeletal tethering and is essential for protein synthesis-dependent synaptic plasticity (Rangaraju et al., 2019).

### Actomyosin in mitochondria and endosome function

The force-generating ability of actin polymerisation and actomyosin contraction is not only important for muscle contractions, cellular forward movement and intracellular pathogen movements via their comet tails, but also for organelle function.

Andrew Moore (HHMI Janelia Research Campus, Ashburn, USA) presented data from his previous research at Erika Holzbaur’s lab, where he discovered F-actin waves in interphase cells. These waves cycle in 15-min intervals and are associated with mitochondria, facilitating mitochondria fission and inhibiting fusion (Moore et al., 2016). Thereby, the F-actin waves enhance mitochondrial network dynamics and mixing.

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Investigating endosomal recycling, Tobias Zech (University of Liverpool, UK) showed that endosomal sorting of EGFR and MT1-MMP requires direct actin binding. The binding of actin to endosomal cargo can counteract ubiquitin signals for their lysosomal sorting, thereby regulating the fate of endocytosed receptors. Endosomal actin is regulated through hepatocyte growth factor-regulated tyrosine kinase substrate (HRS), an ESCRT-0 component, by localising the actin nucleating factor Wiscott–Aldrich syndrome protein and SCAR homologue (WASH) to endosomes (MacDonald et al., 2018). Notably, interfering with actin-mediated recycling of MT1-MMP resulted in defective cancer cell invasion. Staying with the function of actin during endosomal sorting (Wang et al., 2018), Alina Fedoseienko (Mayo Clinic, Rochester, USA) demonstrated that depletion of the recently described COMMD–CCDC22–CCDC93 (CCC) complex results in hyperaccumulation of actin on endosomes.

Next, Felix Straub (University Hospital Regensburg, Germany) described the role of a splice variant of the actin nucleator Spire1 (Pylypenko et al., 2016), Spire1E13, in myosin V-dependent mitochondrial mobility. Folma Buss (University of Cambridge, UK) then showed that myosin VI, which is unique in its ability to move to the minus-ends of actin filaments, is involved in mitochondrial quality control: damaged and ubiquitylated mitochondria recruit myosin VI via complex formation with Parkin, resulting in the formation of F-actin cages. These F-actin cages form a physical barrier, thereby preventing fusion with undamaged mitochondria (Kruppa et al., 2018).

### Actin diversity and organelle movement

Post-translational modification (PTM) of proteins is a key regulatory mechanism of protein function in cells. Tubulin PTMs have recently emerged as pivotal tuners of microtubule-based transport and dynamics, and collectively define a ‘tubulin code’ that governs intricate microtubule functions. In contrast, much less is known about actin PTMs that may coordinate actin cytoskeleton-dependent processes. Harry Higgs (Dartmouth College, Hanover, USA) presented data on INF2, a formin involved in mitochondrial fission and shape changes. In an elegant analysis of chromatographic fractions from mouse brain, he identified cyclase-associated protein (CAP) as forming a complex with lysine-acetylated actin, thus inhibiting INF2 (A et al., 2019). Given the importance of post-translationally modified actin in this and other studies, Harry proposed that there is an ‘actin code’ in analogy with the ‘tubulin code’. Henriette Aksnes (University of Bergen, Norway) presented compelling functional data on N-terminal acetylation of actin, the largest protein modification in cells – nearly 80% of all proteins are N-terminally acetylated. She demonstrated that the N-terminus of both  $\beta$ - and  $\gamma$ -actin is acetylated by NAA80, an enzyme whose loss increases lamellipodia and filopodia numbers, causes cells to migrate faster and fragmentation of the Golgi (Aksnes et al., 2018). By using non-migrating NAA80-knockout cells, she convincingly argued that Golgi fragmentation is independent of hypermotility. Additional diversity in actin populations may come from multiple isoforms of the actin-nucleating complex Arp2/3. Michael Way (The Francis Crick Institute, London, UK) told us ‘the Arp2/3 complex is more complex’ (Abella et al., 2016) and explained that individual Arp2/3 components and their various isoforms can give rise to eight different combinations of Arp2/3 in cells. He then showed that, while complete depletion of Arp2/3 reduced invadopodia, knockdown of some isoforms reduced, and loss of other isoforms unexpectedly increased, the number of invadopodia, suggesting different tasks for different subunits.

### Microtubules and motors

Intracellular transport of cargoes along microtubule tracks is driven by a wide variety of molecular motors. How the microtubule network of cells guides these motors and how different motors contribute to transport are important open questions in the field. Lukas Kapitein (Utrecht University, The Netherlands) summarised how his lab uses light-mediated guidance of motors to probe microtubule track selectivity of kinesin motors in rat hippocampal neurons. His results demonstrated that kinesin-1 prefers stable (acetylated) microtubules whereas kinesin-3 favours dynamic (tyrosinated) microtubules. Importantly, expansion and STED microscopy revealed a unique microtubule network architecture in dendrites wherein tyrosinated and acetylated microtubules are spatially separated and have opposite orientations (Tas et al., 2017). Extending the focus on neuronal transport, Sandra Encalada (The Scripps Research Institute, La Jolla, USA) discussed how prion protein vesicles are transported along axons to the cell surface by microtubule motors (Encalada et al., 2011). After presenting data that showed that the mutant prion protein PrP<sup>PG14</sup> forms aggregates in axons in a kinesin-1-dependent manner, she proposed a model of how the misfolded protein clearance pathway RESET may contribute to the biogenesis of mutant prion protein aggregates. Interestingly, super-resolution and scanning electron microscopy revealed a differential impairment of the various post-translationally modified axonal microtubules, suggesting that aggregates may remodel the microtubule cytoskeleton. Artur Ezquerro (IRB Barcelona, Spain) then expanded the discussion on kinesins by explaining how localisation of the microtubule depolymerising kinesin Kif2A to centrosomes is important for controlling microtubule nucleation. Using super-resolution microscopy and three-dimensional reconstruction, he showed that depletion of Kif2A or its interactor GCP8 (a  $\gamma$ -tubulin ring complex protein) (Teixidó-Travesa et al., 2010) not only stimulates microtubule nucleation but also compromises Golgi integrity. Loss of Kif2A or GCP8 also causes cell cycle exit, which his biochemical data suggested could be due to reduced mTOR activity. Switching direction from kinesins to dynein, Kirsten Garner (University of Manchester, UK) introduced KASH5, a component of meiosis-specific the linker of nucleoskeleton and cytoskeleton (LINC) complexes, as a new adaptor of dynein–dynactin. To understand whether KASH5 can compete with known dynein activators or adaptors, she introduced it into somatic cells where it is normally absent. Interestingly, while cytoplasmic KASH5 disrupts dynein function, at the nuclear envelope, it recruits dynein–dynactin via its N-terminus. Importantly, this function requires dynein light intermediate chains, which the laboratory had previously shown are also needed for spindle assembly (Jones et al., 2014).

### Septins and membrane reorganisation

While much of the conference focussed on the roles of the actin and microtubule cytoskeleton in organelle dynamics, a few of the speakers also discussed the role of septins at sites of membrane reorganisation. One of the outstanding questions in the septin field, namely, how can 32 nm oligomers sense micrometre scale curvatures, was addressed by Amy Gladfelter (University of North Carolina, Chapel Hill, USA). The answer, tested with a series of elegant experiments *in vitro*, is via insertion of the amphipathic helix of Cdc12/Sept6 into the lipid bilayer (Cannon et al., 2019). The spacing of these helices in the septin oligomer contributes to the curvature preference. Roberto Weigert (National Institutes of Health, Bethesda, USA) and Thomas Nightingale (Queen Mary University of London, UK) broadened the discussion

of septins to regulated secretion by presenting data that indicated the importance of complex actin and septin structures in ensuring the release of large granules. Roberto showed elegant work using intravital subcellular microscopy (ISMic) that suggested inward polymerisation of branched actin is required, after an initial recruitment of linear actin, for vesicle incorporation into the plasma membrane (Ebrahim and Weigert, 2019). These actin cages also contain both septin and myosin (Ebrahim et al., 2019). Thomas then demonstrated that the formation of a septin ring precedes actin-ring assembly around the Weibel–Palade body of endothelial cells and that both ring structures are required for efficient release of the cargo von Willebrand factor (Nightingale et al., 2018).

### Exocytosis

In a series of talks on exocytosis, we heard of the diverse mechanisms required for the release of molecules to the plasma membrane and into the extracellular space (van Niel et al., 2018), as well as the transfer of proteins to a neighbouring cell. Graça Raposo (Institut Curie, Paris, France) introduced a pigmentation synapse that is formed by interactions between melanocytes and keratinocytes. These contacts require functional caveolae that act as mechano-signalling sensors that consequently facilitate melanosome transfer. Franck Perez (Institut Curie, Paris, France), then presented data on another form of cell–cell communication, whereby ephrinA1 is passed from one cell to another via cytonemes in an actin- and receptor-dependent manner. By using cargo synchronisation methods, he also showed that there are microtubule-dependent hotspots of exocytosis at focal adhesions (Fourriere et al., 2019).

Microtubules and their motors are implicated in many membrane trafficking processes, and both Anna Akhmanova (Utrecht University, The Netherlands) and Lei Lu (Nanyang Technological University, Singapore) focussed on the role of kinesin and its binding partners in exocytosis. Anna highlighted the importance of going ‘back-to-basics’ to untangle the contribution of kinesins in transporting secretory vesicles. Using a HeLa cell line lacking all four candidate kinesins as a ‘test tube’ for her experiments, she demonstrated functions for different kinesins that otherwise could have been missed due to redundancy. High-resolution imaging showed that Kif5B moves slower and associates with the longer-lived microtubules, whereas Kif13B moves more rapidly and is associated with growing microtubule plus-ends. Lei then discussed two poorly-described Golgi proteins, Mon2 and Dopey-1, which form a complex that binds Kif5B and is required for centrifugal trafficking.

### Golgi

The Golgi is a central hub of cellular trafficking pathways and its structure and function are intimately linked to the cytoskeleton. Using cells plated on micropatterns to generate high-throughput density maps, Kristine Schauer (CNRS, Institut Curie, Paris, France) found a new role for myosin 1C in Golgi organisation that is also important for cargo trafficking (Capmany et al., 2019). Michael Krauss (Leibniz Research Institute of Molecular Pharmacology, Berlin, Germany) similarly highlighted a new function of septin 1 in Golgi integrity and secretion. He showed that septin 1 localises at the Golgi in a GM130-dependent manner where, in concert with Cep170, it is required for efficient microtubule nucleation (Song et al., 2019). The relative contribution of centrosomal versus Golgi-derived microtubules to Golgi integrity was addressed next by Irina Kaverina (Vanderbilt University, Nashville, USA), who has previously shown that proper Golgi assembly requires both microtubule populations

(Vinogradova et al., 2012). She described that cell cycle-dependent changes in Golgi morphology rely on at least one microtubule subpopulation and are important for directional cell motility, and she further speculated on the role of motor activity in these rearrangements. Finally, Jonathan Dragwidge (Heidelberg University, Germany) discussed that the trans-Golgi network (TGN) of plants (Dragwidge et al., 2019) could function as both an early and recycling endosome. He demonstrated that this is achieved by segregating these functions into Rab GTPase-positive TGN sub-domains as well as by dissociating the TGN from the Golgi stack.

### Autophagy

Autophagy requires extensive membrane reorganisation that involves dynamic changes in lipid composition and actin organisation. Dyche Mullins (University of California, San Francisco, USA) addressed the role of different actin networks at the various stages of autophagy. He focussed on JMY, a nucleation promoting factor that, upon starvation, relocates from the nucleus to LC3-positive membranes. The interaction of JMY with LC3 enhances actin nucleation and the formation of comet tails (Hu and Mullins, 2019). Sharon Tooze (The Francis Crick Institute, London, UK) then discussed the initiation of autophagy by presenting elegant data that showed the formation of a unique Atg9 compartment upon starvation. This compartment is formed of multiple vesicles that are enriched in ARFFIP2 and PI4KIII $\beta$ . Its formation and in particular the presence of PI4KIII $\beta$  is required for the conversion of PI to PI4P at the initiation site and for subsequent phagophore formation (Judith et al., 2019). While these talks focussed on the complex series of autophagy events that occur upon starvation, Erika Holzbaur (University of Pennsylvania, Philadelphia, USA) highlighted that, in neurons, autophagy is a homeostatic process (Stavoe and Holzbaur, 2019). She showed that increased levels of stalled autophagosome biogenesis are observed in neurons of old mice, which is partly dependent on the phosphorylation state of WIPI2. She further demonstrated that overexpression of WIPI2 in old neurons increases productive autophagosome formation, while its depletion in young neurons inhibits the rate of autophagosome biogenesis.

### Positioning and squeezing of the nucleus

The nucleus is the largest and stiffest organelle in the cell, requiring cytoskeletal forces for squeezing (e.g. through pores) and intracellular positioning (Calero-Cuenca et al., 2018).

Edgar Gomes (iMM University of Lisbon, Portugal) presented data on novel regulators of nuclear positioning during cell migration in wound-healing scratch assays. These included Ctdnep1/Dullard, which binds to the actin-binding and -bundling protein Eps8L2 and promotes the formation and stabilisation of dorsal actin cables required for nuclear positioning. Interestingly, the phosphatase domain of Ctdnep1/Dullard is not necessary for the binding of Eps8L2, suggesting a unique mechanism to regulate the actin cytoskeleton on the dorsal side of the nucleus, and thus nuclear movement. Allen Liu (University of Michigan, USA) then described the reconstitution of parts of the LINC complex, including full-length SUN proteins, in artificial nuclear membranes using a cell-free expression system (Majumder et al., 2018). This could be a valuable source for future mechanistic studies of the assembly and function of the LINC complex.

Dan Starr (University of California, Davis, USA) presented the establishment of *Caenorhabditis elegans* as a model to investigate nuclear positioning and squeezing *in vivo*. Hypodermal larval P-cell



nuclei have to traverse an extremely narrow space (~5% of their width), and failure of this event results in animals without vulva and motor neurons (Bone et al., 2016). Based on screening approaches, Dan showed a role for TOCA-1, CGEF-1 and a divergent filamentin during P-cell nuclear squeezing *in vivo*. Rebecca McGillivray (University of California, San Francisco, USA) then introduced a non-standard model organism to investigate nuclear positioning and nuclear shape changes: the giant ciliate *Stentor coeruleus* (Sood et al., 2017 preprint) bears a macronucleus in a 'string of beads' shape. During regeneration, the nucleus undergoes massive shape changes through condensation and de-condensation. Rebecca showed that this process depends on CSE-1, known to function during nuclear transport and to be associated with microtubules.

### Membrane contact sites

The first talk covering membrane contacts and lipids was given by Wanda Kukulski (MRC Laboratory of Molecular Biology, Cambridge, UK) who used correlative fluorescence microscopy and electron tomography to demonstrate non-homogenous distribution of proteins that mediate ER contacts at the plasma membrane. She further presented data that revealed the molecular organisation of tricalbins. Her work suggested that tricalbins may serve in previously undescribed cellular lipid distribution pathways (Hoffmann et al., 2019 preprint). Continuing this theme, Magdalena Marek (University of Lausanne, Switzerland) addressed how sterols are moved around in fission yeast cells. Using live imaging and correlative light and electron microscopy, she showed that sterols present in the plasma membrane can be internalised independently of actin polymerisation, via endosomes. The initial stage of sterol internalisation is mediated by the previously uncharacterised protein Ltc1. María Isabel Geli (Institute for Molecular Biology of Barcelona, Spain) then discussed endocytic membrane deformations in budding yeast. Using time-resolved electron microscopy (achieved by integrating live-cell fluorescence microscopy and quantitative immunoelectron microscopy data), she showed that endocytic invaginations become associated with cortical ER at the onset of actin polymerisation (Encinar Del Dedo et al., 2017). At these contact sites, sterols are transferred via Osh2/3 and this is required for recruitment of actin for endocytosis. Michael Schrader (University of Exeter, UK) then switched to peroxisomes, which are organelles essential for cellular redox balance and lipid metabolism. He showed that, unlike in mammalian cells, peroxisomes in the pathogenic fungus *Ustilago maydis* do not move around by recruiting microtubule motors, but instead hitchhike on early endosomes. He also showed that important peroxisome–ER contacts occur through ACBD5–VAP tethers in mammalian cells, which restrict peroxisome movement. Indeed, loss of ACBD5 reduces lipid transfer between the ER and peroxisomes thus impacting peroxisome membrane dynamics (Costello et al., 2017).

### Concluding remarks and future perspectives

A diverse and international group of scientists met in Lisbon with a shared interest in understanding the organelle–cytoskeleton interface. This stimulating meeting combined the fields of membrane organisation, trafficking and the cytoskeleton – and disciplines ranging from molecular cell biology to biophysics. We witnessed the use of classical, as well as less-explored model systems, to address an emerging hot-topic in cell biology – organellar contact sites. The scientific talks were complemented with short sponsor talks on recent developments in holographic (NanoLive, Ecublens, Switzerland) and super-resolution

microscopy (Izasa Scientific, Barcelona, Spain; and ONI, Oxford, UK). In addition, Sharon Ahmad (Journal of Cell Science, Cambridge, UK) shared her personal path to becoming an editor and gave insights into the publication process. Moreover, Anna Akhmanova, Edgar Gomes and Heidi McBride gave 'Career at a glance' talks, sharing anecdotes and career advice with a common theme: follow what is really interesting to you, accept failures, enjoy what you are doing, and enjoy the thrill of discovery!

### References

- A, M., Fung, T. S., Kettenbach, A. N., Chakrabarti, R. and Higgs, H. N. (2019). A complex containing lysine-acetylated actin inhibits the formin INF2. *Nat. Cell Biol.* **21**, 592–602. doi:10.1038/s41556-019-0307-4
- Abella, J. V., Galloni, C., Pernier, J., Barry, D. J., Kjaer, S., Carlier, M.-F. and Way, M. (2016). Isoform diversity in the Arp2/3 complex determines actin filament dynamics. *Nat. Cell Biol.* **18**, 76–86. doi:10.1038/ncb3286
- Aksnes, H., Marie, M., Arnesen, T. and Drazic, A. (2018). Actin polymerization and cell motility are affected by NAA80-mediated posttranslational N-terminal acetylation of actin. *Commun Integr Biol* **11**, e1526572. doi:10.1080/19420889.2018.1526572
- Bone, C. R., Chang, Y.-T., Cain, N. E., Murphy, S. P. and Starr, D. A. (2016). Nuclei migrate through constricted spaces using microtubule motors and actin networks in *C. elegans* hypodermal cells. *Development* **143**, 4193–4202. doi:10.1242/dev.141192
- Calero-Cuenca, F. J., Janota, C. S. and Gomes, E. R. (2018). Dealing with the nucleus during cell migration. *Curr. Opin. Cell Biol.* **50**, 35–41. doi:10.1016/j.ccb.2018.01.014
- Cannon, K. S., Woods, B. L., Crutchley, J. M. and Gladfelter, A. S. (2019). An amphipathic helix enables septins to sense micrometer-scale membrane curvature. *J. Cell Biol.* **218**, 1128–1137. doi:10.1083/jcb.201807211
- Capmany, A., Yoshimura, A., Kerdous, R., Caorsi, V., Lescure, A., Del Nery, E., Coudrier, E., Goud, B. and Schauer, K. (2019). MYO1C stabilizes actin and facilitates the arrival of transport carriers at the Golgi complex. *J. Cell Sci.* **132**. doi:10.1242/jcs.225029
- Costello, J. L., Castro, I. G., Hacker, C., Schrader, T. A., Metz, J., Zeuschner, D., Azadi, A. S., Godinho, L. F., Costina, V., Findeisen, P. et al. (2017). ACBD5 and VAPB mediate membrane associations between peroxisomes and the ER. *J. Cell Biol.* **216**, 331–342. doi:10.1083/jcb.201607055
- Dragwidge, J. M., Scholl, S., Schumacher, K. and Gendall, A. R. (2019). NHX-type Na<sup>+</sup>(K<sup>+</sup>)/H<sup>+</sup> antiporters are required for TGN/EE trafficking and endosomal ion homeostasis in *Arabidopsis thaliana*. *J. Cell Sci.* **132**. doi:10.1242/jcs.226472
- Ebrahim, S. and Weigert, R. (2019). Intravital microscopy in mammalian multicellular organisms. *Curr. Opin. Cell Biol.* **59**, 97–103. doi:10.1016/j.ccb.2019.03.015
- Ebrahim, S., Chen, D., Weiss, W., Malec, L., Ng, Y., Rebustini, I., Krystofiak, E., Hu, L., Liu, J., Masedunskas, A. et al. (2019). Dynamic polyhedral actomyosin lattices remodel micron-scale curved membranes during exocytosis in live mice. *Nat. Cell Biol.* **21**, 933–939. doi:10.1038/s41556-019-0365-7
- Encalada, S. E., Szpankowski, L., Xia, C. H. and Goldstein, L. S. (2011). Stable kinesin and dynein assemblies drive the axonal transport of mammalian prion protein vesicles. *Cell* **144**, 551–565. doi:10.1016/j.cell.2011.01.021
- Encinar Del Dedo, J., Idrissi, F. Z., Fernandez-Golbano, I. M., Garcia, P., Rebollo, Grötsch, H. and Geli, M. I. (2017). ORP-mediated ER contact with endocytic sites facilitates actin polymerization. *Dev. Cell* **43**, 588–602.e6. doi:10.1016/j.devcel.2017.10.031
- Fourriere, L., Kasri, A., Gareil, N., Bardin, S., Bousquet, H., Pereira, D., Perez, F., Goud, B., Boncompain, G. and Miserey-Lenkei, S. (2019). RAB6 and microtubules restrict protein secretion to focal adhesions. *J. Cell Biol.* **218**, 2215–2231. doi:10.1083/jcb.201805002
- Hoffmann, P. C., Bharat, T. A. M., Wozny, M. R., Miller, E. A. and Kukulski, W. (2019). Rod-shaped tricalbins contribute to PM asymmetry at curved ER-PM contact sites. *bioRxiv*, 579128. doi:10.1101/579128
- Hu, X. and Mullins, R. D. (2019). LC3 and STRAP regulate actin filament assembly by JMY during autophagosome formation. *J. Cell Biol.* **218**, 251–266. doi:10.1083/jcb.201802157
- Jones, L. A., Villemant, C., Starborg, T., Salter, A., Goddard, G., Ruane, P., Woodman, P. G., Papalopulu, N., Woolner, S. and Allan, V. J. (2014). Dynein light intermediate chains maintain spindle bipolarity by functioning in centriole cohesion. *J. Cell Biol.* **207**, 499–516. doi:10.1083/jcb.201408025
- Judith, D., Jefferies, H. B. J., Boeing, S., Frith, D., Snijders, A. P. and Tooze, S. A. (2019). ATG9A shapes the forming autophagosome through Arfap12 and phosphatidylinositol 4-kinase IIIβ. *J. Cell Biol.* **218**, 1634–1652. doi:10.1083/jcb.201901115
- Kruppa, A. J., Kishi-Itakura, C., Masters, T. A., Rorbach, J. E., Grice, G. L., Kendrick-Jones, J., Nathan, J. A., Minczuk, M. and Buss, F. (2018). Myosin VI-

- dependent actin cages encapsulate parkin-positive damaged mitochondria. *Dev. Cell* **44**, 484–499. doi:10.1016/j.devcel.2018.01.007
- Kumar, N., Leonzino, M., Hancock-Cerutti, W., Horenkamp, F. A., Li, P., Lees, J. A., Wheeler, H., Reinisch, K. M. and De Camilli, P. (2018). VPS13A and VPS13C are lipid transport proteins differentially localized at ER contact sites. *J. Cell Biol.* **217**, 3625–3639. doi:10.1083/jcb.201807019
- MacDonald, E., Brown, L., Selvais, A., Liu, H., Waring, T., Newman, D., Bithell, J., Grimes, D., Urbé, S., Clague, M. J. et al. (2018). HRS-WASH axis governs actin-mediated endosomal recycling and cell invasion. *J. Cell Biol.* **217**, 2549–2564. doi:10.1083/jcb.201710051
- Majumder, S., Willey, P. T., Denies, M. S., Liu, A. P. and Luxton, G. W. G. (2018). A synthetic biology platform for the reconstitution and mechanistic dissection of LINC complex assembly. *J. Cell Sci.* **132**, jcs219451. doi:10.1242/jcs.219451
- Mehta, K., Chacko, L. A., Chug, M. K., Jhunjhunwala, S. and Ananthanarayanan, V. (2019). Association of mitochondria with microtubules inhibits mitochondrial fission by precluding assembly of the fission protein Dnm1. *J. Biol. Chem.* **294**, 3385–3396. doi:10.1074/jbc.RA118.006799
- Moore, A. S., Wong, Y. C., Simpson, C. L. and Holzbaur, E. L. (2016). Dynamic actin cycling through mitochondrial subpopulations locally regulates the fission-fusion balance within mitochondrial networks. *Nat. Commun.* **7**, 12886. doi:10.1038/ncomms12886
- Nightingale, T. D., McCormack, J. J., Grimes, W., Robinson, C., Lopes da Silva, M., White, I. J., Vaughan, A., Cramer, L. P. and Cutler, D. F. (2018). Tuning the endothelial response: differential release of exocytic cargos from Weibel-Palade bodies. *J. Thromb. Haemost.* **16**, 1873–1886. doi:10.1111/jth.14218
- Peterka, M. and Kornmann, B. (2019). Miro-dependent mitochondrial pool of CENP-F and its farnesylated C-terminal domain are dispensable for normal development in mice. *PLoS Genet.* **15**, e1008050. doi:10.1371/journal.pgen.1008050
- Pylypenko, O., Welz, T., Tittel, J., Kollmar, M., Chardon, F., Malherbe, G., Weiss, S., Michel, C. I., Samol-Wolf, A., Grasskamp, A. T. et al. (2016). Coordinated recruitment of Spir actin nucleators and myosin V motors to Rab11 vesicle membranes. *Elife* **5**, e17523. doi:10.7554/eLife.17523
- Rangaraju, V., Lauterbach, M. and Schuman, E. M. (2019). Spatially stable mitochondrial compartments fuel local translation during plasticity. *Cell* **176**, 73–84.e15. doi:10.1016/j.cell.2018.12.013
- Song, K., Gras, C., Capin, G., Gimber, N., Lehmann, M., Mohd, S., Puchkov, D., Rödigier, M., Wilhelmi, I., Daumke, O. et al. (2019). A SEPT1-based scaffold is required for Golgi integrity and function. *J. Cell Sci.* **132**, jcs225557. doi:10.1242/jcs.225557
- Sood, P., McGillivray, R. and Marshall, W. F. (2017). The Transcriptional program of regeneration in the giant single cell, stentor coeruleus. *bioRxiv*, 240788. doi:10.1101/240788
- Stavoe, A. K. H. and Holzbaur, E. L. F. (2019). Axonal autophagy: mini-review for autophagy in the CNS. *Neurosci. Lett.* **697**, 17–23. doi:10.1016/j.neulet.2018.03.025
- Sugiura, A., McLelland, G.-L., Fon, E. A. and McBride, H. M. (2014). A new pathway for mitochondrial quality control: mitochondrial-derived vesicles. *EMBO J.* **33**, 2142–2156. doi:10.15252/embj.201488104
- Tas, R. P., Chazeau, A., Cloin, B. M. C., Lambers, M. L. A., Hoogenraad, C. C. and Kapitein, L. C. (2017). Differentiation between oppositely oriented microtubules controls polarized neuronal transport. *Neuron* **96**, 1264–71.e5. doi:10.1016/j.neuron.2017.11.018
- Teixidó-Travesa, N., Villén, J., Lacasa, C., Bertran, M. T., Archinti, M., Gygi, S. P., Caelles, C., Roig, J., Lüders, J. and Bettencourt-Dias, M. (2010). The gammaTuRC revisited: a comparative analysis of interphase and mitotic human gammaTuRC redefines the set of core components and identifies the novel subunit GCP8. *Mol. Biol. Cell* **21**, 3963–3972. doi:10.1091/mbc.e10-05-0408
- Valm, A. M., Cohen, S., Legant, W. R., Melunis, J., Hershberg, U., Wait, E., Cohen, A. R., Davidson, M. W., Betzig, E. and Lippincott-Schwartz, J. (2017). Applying systems-level spectral imaging and analysis to reveal the organelle interactome. *Nature* **546**, 162–167. doi:10.1038/nature22369
- Van Niel, G., D'angelo, G. and Raposo, G. (2018). Shedding light on the cell biology of extracellular vesicles. *Nat. Rev. Mol. Cell Biol.* **19**, 213–228. doi:10.1038/nrm.2017.125
- Vinogradova, T., Paul, R., Grimaldi, A. D., Loncarek, J., Miller, P. M., Yampolsky, D., Magidson, V., Khodjakov, A., Mogilner, A., Kaverina, I. et al. (2012). Concerted effort of centrosomal and Golgi-derived microtubules is required for proper Golgi complex assembly but not for maintenance. *Mol. Biol. Cell* **23**, 820–833. doi:10.1091/mbc.e11-06-0550
- Wang, J., Fedoseienko, A., Chen, B., Burstein, E., Jia, D. and Billadeau, D. D. (2018). Endosomal receptor trafficking: Retromer and beyond. *Traffic* **19**, 578–590. doi:10.1111/tra.12574